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ATROPHY OF THE CEREBELLUM IN A CAT.

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THE study, which supplies the material for this article, was made in the Neurological Laboratory of Clark University, and under the direction of Professor H. H. Donaldson. The specimen which forms the basis of this study was kindly furnished by Dr. B. R. Benner, of Lowell, Mass., and to him we are indebted for a description of the cat before it was brought to the university laboratory.

Dr. Benner informs us that the cat suddenly became paralyzed when three months old. He was observed coming out of the stable one morning apparently as well as ever, but a very short time after it was noticed that his hind legs had become almost entirely helpless. For several days he was disinclined to eat, and all attempts at locomotion were accompanied by spasmodic jerkings. In the act of walking, the hinder parts seemed to get along considerably faster than the fore limbs, and faster than he desired, giving a ludicrous effect. After a time he recovered his appetite, became cheerful and lively, continued to improve, and, with the exception of the permanent disability to the limbs, he seemed to be as well as ever, for his development was not retarded, as

he grew to normal size. During the five months previous to his being brought to the laboratory there was no change manifested.

There were but two kittens born in the litter, this cat and a sister. The latter is living and is bright and healthy. In disposition there was a slight difference, in that the subject was more shy and timid than the healthy sister, running away more quickly on the approach of strangers. Sexually, he was incompetent, his efforts in this direction being blundering and abortive. There is no history of any injury whatever; yet the trouble manifested itself suddenly, and was not progressive. Both sides were affected and were so from the very first.

When brought to the laboratory, the cat was eleven months old. Here it was observed that he was timid and shy; well nourished; could walk and trot; but his gait was zigzag and staggering, as his hind legs spread somewhat laterally in locomotion; all four legs appeared somewhat ataxic, but hind legs more so; inco-ordination of the limbs, especially of the hind legs, which were raised very high and placed so far forward that they overstepped the forelegs in walking; peculiar movements most marked when going slowly; could jump off a stool, also raise himself on his hind legs, but manifested a disinclination toward doing so; volitional movements of the head and neck spasmodic and jerky; he trembled a great deal all the time, but especially after moving about; his sitting posture normal; shook a good deal when turning around to look out of the window; eyes also normal; tail rigid; hind legs seemed a trifle thin; miowed and purred.

The results of the autopsy were negative save in the case of the cerebellum, which was slightly smaller and more resistant to the knife than the normal, and seemed shrunken, the cortex appearing rather thin; fore-brain in normal condition; appeared well nourished; viscera in good condition; bladder full and distended; spinal cord normal; some fat in canal; nothing to be seen in sciatic nerves. The cerebellum and spinal cord, as well

as one lumbar and one dorsal spinal ganglion, were preserved for further examination and study.

These portions were first placed in a $2\frac{1}{2}$ per cent. solution of bichromate of potash, plus $\frac{1}{8}$ its volume of 95 per cent. of alcohol, for the purpose of hardening, where they were kept for six weeks, when they were washed in water for thirty-six hours, put in 95 per cent. alcohol for one day, and then kept for a time in 80 per cent. alcohol. The cerebellum was cut in halves by a section passing through the vermis. Each half was considered as composed of three divisions which resulted from dividing each half by two more sections in the same plane and at equal intervals from one another. Each division, therefore, represented a slice of the cerebellum about four millimeters in thickness. The slice nearest the middle line was divided into a dorso-cephalic portion, designated as C, and a ventro-caudal portion, A. The second slice of each half was also divided into two portions, a dorso-cephalic, D, and a ventro-caudal, B. The remaining portion, E, was undivided, and represented the lateral third of the hemisphere. These portions were imbedded in celloidin in the usual manner, and then cut with the microtome into sections varying from five to six one-hundredths of a millimeter in thickness. The method of staining was as follows: The three sections were first washed in water, and then placed in picro-fuchsin (Van Gieson), where they were allowed to remain two minutes. The picro-fuchsin was made from a saturated solution of picric acid, which was colored a garnet red by adding an aqueous solution of acid fuchsin. On being taken from the picro-fuchsin, the section was washed in 95 per cent. alcohol, and then placed in Delafield's hæmatoxylin for one minute. It was then washed in water, after which it was placed in a solution of lithium carbonate for about a minute. It was again washed in 95 per cent. alcohol, and then allowed to lie in oil (*origani cretici*) for a time, when it was mounted in the usual manner.

For the purpose of comparison, four normal cerebel-

lums were taken to serve as controls. Controls I., II., and III. indicate the cerebellums of three cats not related, three months old, selected because the cat whose cerebellum forms the basis our study, was three months old when the ataxic symptoms were first manifested, and it was therefore desirable to secure specimens of the cortex in animals of that age for the initial comparison. Control IV. represents the cerebellum of a six months cat. The cerebellum of each of these four controls was cut as described above in the case of the ataxia, except that the slice nearest the middle line was, in the case of the four controls, cut into three divisions, A, C, and F, instead of two as in the ataxic. That portion of the control indicated by F, is included in A of the ataxic.

As a preparation for measurement, outlines were drawn of each of the sections of all the controls, as well as the ataxic specimen. In each section, four regions, not contiguous, were indicated in the drawing, and these were then measured in the mounted specimen; the measuring being for the purpose of determining the thickness of the molecular layer, and the order or method of measurement, in determining the thickness of this molecular layer was this: First, at the summit of the folium; second, at one side of the sulcus (side A) midway between the summit and base; third, at a corresponding point on the opposite side of the sulcus (side B), and lastly, at the base of the sulcus between two folii. Thus, to make a *résumé*, we have four normal specimens, which, for the purpose of comparison, serve as controls, and one ataxic specimen. Each control is cut into twelve divisions (six divisions in each half), and the ataxic specimen is cut into ten. Four measurements (summit, two sides, and base) are made at the four different localities in each section, making a total of 928 measurements. The results of these measurements, which, as has been stated, were made with a view to determine the thickness of the molecular layer in the four controls, as well as in the ataxic specimen, are comprehended in the following table: The measurements were made with the Zeiss apochromatic

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objective 16, and compensating eye-piece 6. These measurements are therefore expressed in thousandths of a millimeter. The limits of measurement were the bases of the Purkinje cells and the inner surface of the pia.

TABLE I.—SHOWING THICKNESS OF MOLECULAR LAYER AT FOUR LOCALITIES IN EACH PORTION OF THE FOUR CONTROLS AND THE ATAXIC SPECIMEN. THE UNIT OF MEASURE IS THE MILLIMETER.

RIGHT HALF.							LEFT HALF.						
	PORTION.	SUMMIT.	SIDE A.	SIDE B.	BASE.	AVER.	SUMMIT.	SIDE A.	SIDE B.	BASE.	AVER.	TOTAL AVER.	
CONTROL I.	A	.224	.316	.308	.280	.282	.316	.276	.292	.316	.300	.291	
	B	.264	.268	.288	.312	.283	.288	.292	.296	.324	.300	.291	
	C	.264	.292	.308	.288	.288	.232	.292	.312	.300	.284	.286	
	D	.264	.248	.292	.268	.268	.240	.316	.288	.324	.292	.280	
	E	.272	.312	.304	.332	.305	.268	.324	.380	.357	.332	.318	
	F	.204	.236	.248	.296	.246	.248	.288	.280	.288	.276	.261	
	Aver.	.248	.278	.291	.296	.278	.265	.298	.308	.318	.297	.287	
CONTROL II.	A	.236	.308	.280	.304	.282	.320	.268	.284	.300	.293	.287	
	B	.272	.340	.292	.316	.305	.288	.288	.312	.300	.297	.301	
	C	.224	.284	.256	.308	.268	.272	.260	.272	.312	.279	.273	
	D	.256	.288	.280	.300	.281	.316	.312	.280	.312	.305	.293	
	E	.264	.264	.288	.315	.282	.272	.316	.324	.336	.312	.297	
	F	.232	.256	.276	.260	.256	.260	.276	.288	.304	.282	.269	
	Aver.	.247	.290	.278	.300	.279	.288	.286	.293	.310	.294	.286	
CONTROL III.	A	.256	.308	.304	.272	.285	.324	.304	.288	.312	.307	.296	
	B	.296	.304	.296	.348	.336	.264	.272	.248	.308	.273	.304	
	C	.232	.256	.256	.268	.253	.264	.308	.288	.272	.283	.268	
	D	.236	.252	.280	.280	.262	.288	.280	.324	.348	.310	.286	
	E	.244	.264	.280	.320	.277	.280	.300	.340	.320	.310	.293	
	F	.252	.248	.252	.280	.258	.268	.304	.312	.304	.297	.277	
	Aver.	.252	.272	.278	.294	.278	.281	.294	.300	.310	.296	.287	
Total	Aver.	.249	.280	.282	.296	.278	.278	.292	.300	.312	.295	.286	
CONTROL IV.	A	.304	.364	.356	.348	.343	.348	.352	.324	.304	.332	.337	
	B	.328	.284	.276	.324	.303	.352	.344	.340	.356	.348	.325	
	C	.336	.324	.284	.356	.325	.308	.336	.304	.304	.313	.319	
	D	.304	.300	.304	.304	.303	.340	.388	.360	.396	.371	.337	
	E	.328	.320	.308	.364	.330	.405	.363	.373	.400	.385	.357	
	F	.320	.316	.304	.348	.322	.324	.308	.332	.332	.324	.323	
	Aver.	.320	.318	.305	.340	.321	.346	.348	.338	.348	.345	.333	
ATAXIC. II Mos.	A	.164	.172	.216	.204	.189	.144	.168	.168	.188	.167	.178	
	B	.172	.176	.164	.180	.173	.180	.184	.200	.220	.196	.184	
	C	.144	.180	.168	.212	.176	.144	.168	.156	.208	.169	.172	
	D	.164	.168	.176	.270	.194	.148	.204	.184	.188	.181	.187	
	E	.368	.340	.328	.358	.351	.272	.232	.244	.288	.259	.305	
	F	.202	.207	.210	.246	.180	.177	.191	.190	.218	.162	.171	
	Aver.	.202	.207	.210	.246	.180	.177	.191	.190	.218	.162	.171	

These measurements show that the atrophy involved all parts of the affected cerebellum except E right. The

nature of the disturbances found in these portions are now to be described with more or less detail.

I.—PIA AND BLOOD-VESSELS.

In the abnormal portions of the ataxic specimen the blood globules do not show so distinctly as in the normal portions of the same specimen, or in the controls. In these same abnormal portions, the pia is somewhat thinner and less regular in its thickness than in the specimens which served as controls. This is especially true with reference to the folds of the pia in the sulci between the folii where, as a rule, the pia is less compact. The walls of the blood-vessels are less clearly defined than in the normal, and contain fewer nuclei, which are abundant in the blood-vessels of the less affected portions.

II.—HISTOLOGY OF THE MOLECULAR LAYER.

The small thread-like fibres (radial fibres) in this layer, most of which run at right angles to the surface, show much more clearly in the ataxic than in the normal, giving to this layer (in the ataxic) a marked striate appearance. Indeed, in the normal specimen, the course of the connective-tissue fibres cannot be traced, while in the diseased portions of the ataxic specimen, one can follow them as far as the Purkinje cell layer, to which their course is direct. They run parallel to each other and without dividing. At their peripheral ending, just at the pia, a few of these fibres branch, but the majority end with pyramidal bases, the broad ends of which abut against the inner surface of the pia. The Purkinje cell processes, which are much thicker than the other fibres, branch and spread, and cannot be traced to the periphery, but lose themselves before reaching the pia. These processes always divide at a short distance from the Purkinje cells, and this division occurs at a greater distance from the cell at the summit of the folii than in the region at the base of the valleys. Throughout the entire molecular layer are distributed small cells which

show their nuclei very clearly. This distribution is without any order, except that the cells are more abundant in the regions contiguous to the Purkinje cell processes and near the pia at the periphery. In the normal portion of the ataxic specimen and in each of the controls, the nuclei of these cells are surrounded by a well-marked cell body, which disappears entirely in the diseased portions, leaving nothing but the bare nuclei remaining. It should also be mentioned that some of these small cells send off very fine processes, which are visible only in the normal. The processes of the Purkinje cells show much more plainly, are longer, and have more branches in the normal than in the ataxic specimen. In the diseased portions, no Purkinje cell processes are to be seen. In no case have the processes from one Purkinje cell been found to come in contact or unite with those of another Purkinje cell; they always remain distinct, even in case of the very finest processes. In the vicinity of the Purkinje cells are a few fibres that run parallel to the surface and are visible only in the ataxic specimen.

III.—THE PURKINJE CELLS.

In the abnormal portions of the ataxic specimen, some of the Purkinje cells have disappeared entirely, leaving open and empty places, but for the most part the cells are only considerably shrunken. It seems that these Purkinje cells are less affected, *i. e.*, they preserve their shape, size, and general character better in the portion of the folii near the base of the sulci than at the summits of the folii. In the ataxic, the Purkinje cells in the affected portion have lost their large round nuclei, which, with their nucleoli, show so plainly in the normal. In the normal these Purkinje cells are very similar in size and shape, while in the ataxic, there is no regularity in this respect. In all the specimens which served as controls, these Purkinje cells were found to be much more numerous at the summits of the cerebellum, where they are packed closely together, than at the base

of the valleys, where they stand quite a distance apart. Small fibres extending toward the granular layer can be seen at their basal end, but cannot be followed save for a very short distance. One of the most marked differences between the tissue of the normal and ataxic specimens is that which comes out in an examination of the Purkinje cells with reference to their size, shape, and general appearance. In the normal portions of the ataxic specimen, where these cells are more like the normal, the radial connective-tissue fibres are less evident. In the degeneration of the Purkinje cells, the processes more remote from the cells are the first to be affected, then the degeneration approaches the cell itself. It begins to shrink and loses its nucleus with its shining nucleolus, as well as its elliptical shape, becoming more and more round as it becomes smaller, until it disappears altogether. All stages of degeneration were observed in the Purkinje cells of the ataxic specimen.

IV.—THE GRANULAR LAYER.

In the normal specimens the granules in the region adjacent to the Purkinje cells are larger than the others, but no structural differences are displayed. The layer of granules is always thickest at the summits of the folii, and thinnest at the bottom. There is no regular arrangement in their distribution, save that where thickest they are found in small groups. We were unable to detect any difference in the appearance of the granular layer of the ataxic specimen as compared with that of the normal, save that in the affected portions of the ataxic, the line of demarcation between this layer and the Purkinje cells is less clearly drawn, and that also in the ataxic, the granules near the Purkinje cells are oftentimes more angular in shape and, on the whole, less closely packed and less numerous in the ataxic than in the normal. The medullary fibres from the central white portion form a network throughout the whole of the granular layer.

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V.—THE WHITE MEDULLARY SUBSTANCE.

There is no difference discernible between the ataxic and normal specimens with reference to the appearance of this white medullary substance.

VI.—CONDITION OF THE SPINAL CORD.

A few scattered degenerated fibres were found in the column of Burdach, especially in the cervical and lumbar regions, but these are restricted and apparently local, since they could not be followed in the thoracic cord.

There was no trace found of any inflammatory process, or any sort of hemorrhage, and the symptoms manifested by the cat were such that, when taken with the thinning and arrested development of the cortex, we are led to describe the disease as a case of simple atrophy, which, of course, is not an explanation. As already intimated, certain degenerations were found in the spinal cord, but in the main study these were not followed out in detail.

GENERAL CONCLUSIONS.

We wish to call attention to the following:

(1) The general fact, so well sustained by the measurements recorded in the table given above, that in the normal specimens the molecular layer of the left hemisphere of the cerebellum is thicker than that of the right hemisphere, is brought out by the heavier figures. By consulting the table it is seen that in only five instances was there a preponderating thickness of this layer on the right half, and since in the characteristic cat's cerebellum, the vermis is turned rather to the right of the middle line, these exceptions might readily be accounted for.

(2) A marked growth in the thickness of the molecular layer the six months' cat as over against the one three months old, is also indicated in the above table. Thus the average thickness of the entire molecular layer in the case of the three months' cat is about .286 of a

millimeter, while that of the six months' cat is .333 of a millimeter, an increase of $16\frac{1}{3}$ per cent.

(3) The relative thickness of the molecular layer at the summits, sides, and bases of the same folium. By consulting the table it is found that the average thickness of the summits in each of the four controls is .248, .247, .252, and .320 respectively, on the right hemisphere, and on the left, .265, .288, .281, and .346, respectively, while the bases taken in the same order exhibit these figures: .296, .300, .294, .340, for the right, and .318, .310, .310, and .348 for the left hemisphere, an increase of 12 per cent. in favor of the regions at the bottom of the fissures. This is just the reverse of the relation that obtains in the cerebrum,

(4) It will be remembered that the ataxic was three months old when it began to manifest symptoms of disease. But by reference to the table, it is seen that the molecular layer is much thinner in the case of the ataxic cat than in the normal three months' cat. We append a table showing this in per cents.

TABLE II.—Showing in per cents. the average thickness of the molecular layer in the diseased cat, accompanied with the average thickness of the same layer in the normal cat three months of age.

REGION.	RIGHT.	LEFT.	TOTAL.
A	55.1 %	50.3 %	52.8 %
B	57.1 %	56.3 %	56.6 %
C	54.2 %	54.0 %	54.0 %
D	64.0 %	44.8 %	55.2 %
E	106.4 %	67.3 %	85.4 %

“Arrested development” alone cannot account for this. It is only explained by an active atrophy.

(5) The distribution of the Purkinje cells. It is an interesting fact in every normal specimen examined, the

Purkinje cells were closely packed together at the summits of the convolutions, where they were found to be very numerous, while at the sides, as we passed toward the base of the sulcus, the distance between these cells was found to increase, and at the very bottom they stood quite far apart. This makes a direct proportion between the thickness of the granular layer and the number of Purkinje cells, as has been already suggested by others.

(6) Certain histological characteristics. (a) Some sort of relation exists between the peripheral processes of the Purkinje cells and the small cells of the molecular layer, but this is only of a general nature. (b) In the degeneration of the Purkinje cells, the processes more remote from the cells are the first to be attacked, then this degeneration approaches nearer and nearer to the cell-body, which begins to shrink, losing its nucleus, becoming smaller and smaller, until it drops out altogether leaving an empty cavity.

It was with the hope of contributing something to comparative pathology that these observations were made.

WORCESTER, MASS., June 28, 1892.

HYSTERIC MUTISM CURED BY FARADISATION.

At a recent meeting of the "Société Médicale des Hôpitaux," Troisier spoke of a case he had presented at a previous meeting of a patient who was suffering from hysterical mutism, but who was now completely cured, after having had five electrical séances with the employment of faradism. The affection had come on abruptly after the patient had suffered some days from dysphasia, and a sort of tardiness in speech, but no real stammering, and it disappeared with equal abruptness after the last application of electricity.

J. C.